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# “Full Plastic-Jacket”: Reconstruction of 18 cm of coronary arteries with bioresorbable vascular scaffolds in a young patient with ST-elevation myocardial infarction and multivessel disease

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The Absorb everolimus-eluting bioresorbable vascular scaffold (Absorb BRS; Abbott Vascular, Santa Clara, CA, USA) promises vascular repair maintaining lumen diameter and restoring vasomotion thereby reducing plaque volume. This technique allows an option for bypass surgery thereafter (“leaving nothing behind”) [1]. Concerns arose from some trials and registries reporting higher rates of stent thrombosis and restenosis, although data are not unequivocal [2, 3]. So far, randomized controlled trials with BRS addressed simple and intermediate lesions. Whether BRS are also suited for more complex lesions is less known [4]. However, from a theoretical point of view, patients with more complex and especially long or multiple lesions may benefit most in the long-term from a therapy with BRS. The presented case was of a young patient with ST-elevation myocardial infarction (STEMI) undergoing multiple percutaneous coronary interventions (PCI) and BRS implantation.

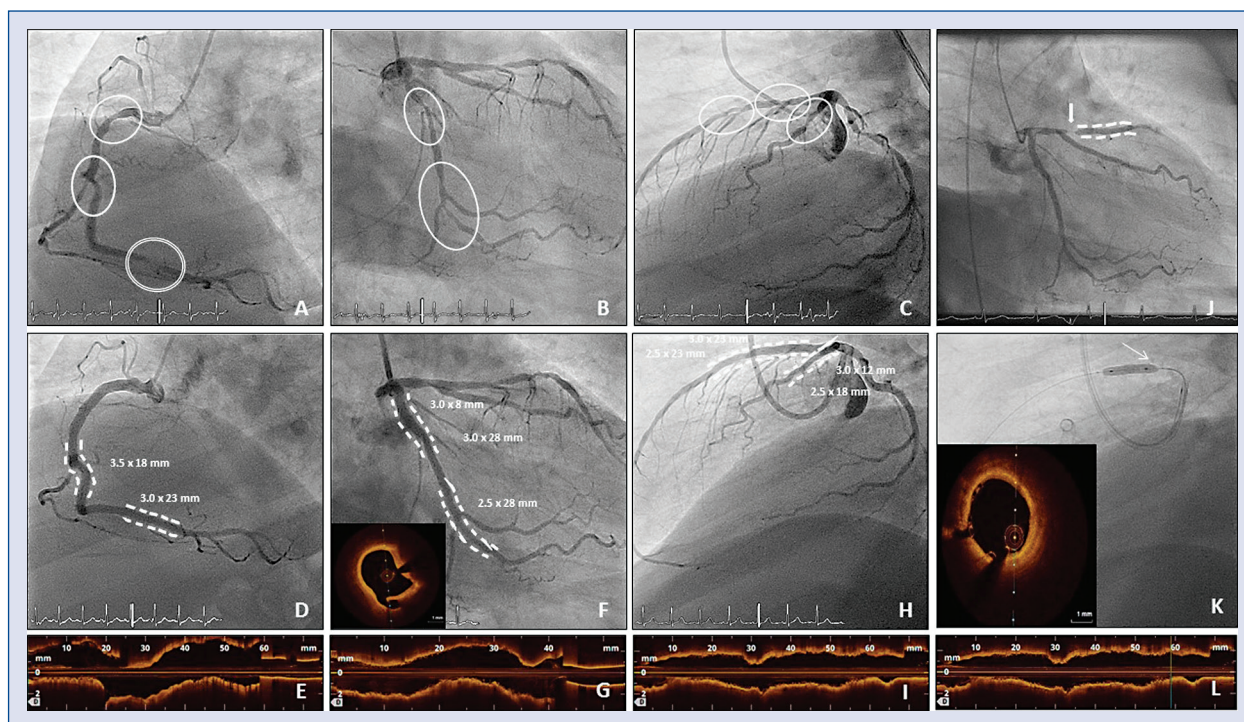
A 55-year-old man was admitted to our hospital with an inferior STEMI, with peak high sensitivity-troponin (hsT) of 5333 ng/L and creatine kinase of 2131 U/L (normal value respectively < 14 ng/L and < 190 U/L). Coronary angiography revealed diffuse triple-vessel disease with subtotal occlusion of the distal right coronary artery (RCA,

Fig. 1A, white double circle), long significant lesions of the left circumflex artery (LCX) and marginal branch (Fig. 1B) as well as severe stenosis of the left anterior descending artery (LAD) and the first diagonal branch (D1) (Fig. 1C). Left ventricular ejection fraction was normal with inferior hypokinesia. First, a 3.0 × 23 mm everolimus-eluting bioresorbable vascular scaffold (BRS, Absorb<sup>®</sup>, Abbott Vascular, Santa Clara, CA, USA) was implanted in the distal RCA (“culprit lesion”) followed by a 3.5 × 18 mm BRS in the mid RCA. Proximally to the latter, a drug-eluting stent (Xience Alpine 4.0 × 28 mm, Abbott) was implanted due to the large vessel size (Fig. 1D), with no overlapping of the previous implanted BRS and no signs of dissection at the optical coherence tomography (OCT) control (Fig. 1E). The patient refused surgical revascularization of the remaining lesions and the residual SYNTAX Score after PCI was 28. Therefore, the following day, the marginal branch and proximal LCX were stented again using two BRS (2.5 × 28 mm and 3.0 × 28 mm) (Fig. 1F). Fluoroscopy showed a proximal-edge dissection, and the OCT confirmed a 4 mm long dissection (insert in Fig. 1F and Fig. 1G; **Supplemental Video 1 — see journal website**) that was treated with a 3.0 × 8 mm BRS with optimal result (**Supplemental Video 2 — see journal website**). After 1 month,

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**Figure 1.** Multiple percutaneous coronary interventions and bioresorbable vascular scaffold (BRS) implantation in a young patient with acute coronary syndrome (ACS); **A, B, C.** Coronary angiography showed a diffuse triple-vessel disease with subtotal occlusion of the distal right coronary artery (RCA, **A**, white double circle), long significant lesions of the left circumflex artery (LCX) and marginal branch (**B**, white circles) as well as severe stenosis of the left anterior descending artery (LAD) and the first diagonal branch (D1) (**C**, white circles); **D.** Percutaneous coronary intervention (PCI) of the RCA: percutaneous transluminal coronary angioplasty (PTCA)/stenting of the distal RCA ("culprit lesion") with a  $3.0 \times 23$  mm everolimus-eluting bioresorbable vascular scaffold (BRS, Absorb<sup>®</sup>, Abbott Vascular, Santa Clara, CA, USA) and of the mid RCA with a  $3.5 \times 18$  mm BRS (the dotted white lines indicate the BRS); **E.** No signs of dissection at the optical coherence tomography (OCT) control; **F and insert, G.** PCI of the LCX on the following day with PTCA/stenting of the marginal branch and of the proximal LCX with two BRS ( $2.5 \times 28$  mm and  $3.0 \times 28$  mm, indicated by the dotted white lines). Control fluoroscopy showed a proximal-edge dissection, which was confirmed by OCT, that was treated with a  $3.0 \times 8$  mm BRS; **H, I.** Control coronary angiography at 1 month. PCI of the mid-LAD ( $3.0 \times 23$  mm and  $2.5 \times 23$  mm BRS, dotted white lines) and of the D1 ( $3.0 \times 12$  mm and  $2.5 \times 18$  mm BRS, dotted white lines). No evidence of dissection at the following OCT control; **J.** Mid-LAD stenosis after the D1 in the context of a non-ST-elevation myocardial infarction-ACS after 3 months (the white arrow indicates the stenosis); **K and insert.** Re-analysis of the previously performed final angiography and OCT of the respecting BRS suggested as an underlying mechanism of this stenosis outside the scaffold, probably a micro-injury of the intima ("geographical missing") due to the recommended post-dilatation (in our case a  $3.0 \times 15$  mm non-compliant balloon at 24 bar); **L.** Longitudinal OCT with no signs of dissection.

control coronary angiography showed an optimal result after PCI. At that time four additional BRS were implanted in the mid-LAD ( $3.0 \times 23$  mm and  $2.5 \times 23$  mm) and in the D1 ( $3.0 \times 12$  mm and  $2.5 \times 18$  mm) (Fig. 1H) with no evidence of dissection at the following OCT control (Fig. 1; [Supplemental Video 3 — see journal web-site](#)). Lesion preparation was always performed using compliant balloon with increasing diameter whereas needed, while post-dilatation was always carried out using non-compliant balloon with high

inflation pressure. 181 mm-BRS-implantation achieved a full revascularization.

After 3 months the patient was again admitted to our hospital with an acute non-STEMI with a peak hsT of 569 ng/L and creatine kinase of 193 U/L. Coronary angiogram revealed a mid-proximal LAD stenosis (Fig. 1J) adjacent to the proximal LAD BRS (scaffold restenosis, according to the current definition within 5 mm proximal and distal to the stent/scaffold [5]). The lesion was treated with a sirolimus drug-eluting stent. Re-analysis

of the previously performed final angiography and OCT of the respecting BRS showed no signs of strut malapposition, edge dissection or thrombus formation. Therefore, the underlying mechanism of this stenosis outside the scaffold was most likely related to the recommended post-dilatation (in our case a  $3.0 \times 15$  mm non-compliant balloon at 24 bar) when using BRS with consequent micro-injury of the intima (“geographical miss”) (Fig. 1K and insert, Fig. 1L) rather than a “classical scaffold stenosis”.

“Full Plastic-Jacket” using BRS is feasible and could be an option for patients with a diffuse disease, however, future studies are necessary to prove long-term benefit of this new therapeutic option [6]. BRS implantation in STEMI patients represents a potential option with a high procedural success rate [7]. Nevertheless, due to lesion preparation and post-dilatation, which are mandatory to achieve an optimal result with BRS [8], there might be a potentially higher risk of early de novo stenosis or scaffold stenosis adjacent or outside the BRS.

**Conflict of interest:** None declared

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